ed in Oppenheim's Clinic, in which special symptoms were present. In several cases the so-called "false foot-tremor" was seen. This is brought out by placing the foot in dorsal flexion, and holding it for several seconds. Typical tremor ensues which differs from the ordinary foot-clonus in that the movements are slower, and less rhythmical, and are seemingly produced by the extensor muscles. The symptom is of value in cases in which the tremor does not appear early, and in cases in which the differentiation between paralysis agitans and senile tremor is difficult. The writer also calls attention to the occurrence of associated movements in cases of shaking palsy. Two cases of unilateral disease are reported, in which active movements of the muscles of the affected side were accompanied by similar movements of the unaffected, although active movements of the latter were followed by no similar state in the diseased areas. In one of these cases the author notes that the side of the face corresponding with the affected half of the body was involved in the rigidity and tremor of the disease. Another case is reported in which there were marked disturbances of sensibility, including diminution of both painful and temperature senses.

The paper concludes with the outline of two cases in which the differential diagnosis between paralysis agitans and senile tremor of arterio-sclerosis was in doubt, and the symptoms pointed again to a strong inter-relation in the pathology of these two diseases. One patient presented the typical manifestations of paralysis agitans, and in addition, headache, vertigo, bulbar manifestations and exaggerated knee-jerk. The second case presented only a superficial resemblance to paralysis agitans, with the exception of a tremor. There was predominance of symptoms particularly suggestive of arteriosclerosis, as paresis of the extremities, spasms, and increase of the patellar tendon reflex and true ankle-clonus, together with mental defects, dysarthria and dysphagia. Cases have been reported in which these manifestations of senile degeneration have faded away, and have been followed by manifestations difficult to distinguish from those of paralysis agitans. Such a variety of symptoms, which point unmistakably to involvement of the cerebral hemispheres, bears strong evidence of an association of this disease with vascular degenerations. JELLIFFE.

CONTRIBUTION A L'ÉTUDE DE L'ANATOMIE PATHOLOGIQUE DE L'HEMIANOPSIE D'ORIGINE INTRA-CÉRÉBRALE (Contribution to the Pathological Anatomy of Hemianopsia of Intra-cerebral Origin). Joukowsky (Nouvelle Iconographie de la Salpêtrière, No. 1, 14th Year, Jan.-Feby., 1901, p. 1).

A study of two cases of hemianopsia caused by the destruction of the cortical visual center. Case I. An old man with left homonymous hemianopsia. He had no paralysis nor sensory aphasia. Autopsy showed a softening in the right hemisphere in the region supplied by the posterior cerebral artery, which had destroyed the region of the calcarine fissure and the neighboring part of the occipital pole. Small softenings were present in the convultions O1 and O2. Histological study showed that the lesion was located in the calcarine fissure, destroying chiefly the lingual and fusiform lobes, and that this lesion penetrated in the adjacent white substance to the internal wall of the posterior horn. This lesion was followed by a sclerosis of the radiate fibers of Gratiolet, by sclerosis of the inferior portion of the retro-lenticular internal capsule, and by that of the inferior posterior part of the optic tract. This sclerosis did not show secondary degeneration. In this case then there is a lesion exclusively of the intra-cerebral visual apparatus, causing the phenomena of hemianopsia. Case II. Double hemianopsia with preservation of the central portion of the field of vision. Autopsy: The right hemisphere presents an old focus of softening upon its inferior surface. The hippocampal convolution is softened, especially in the external posterior half. The retro-lenticular fold, the lingual lobe, the fusiform and the third temporal are all affected. Upon the internal surface, the calcarine fissure is involved in its whole extent. In the left hemisphere there is a softening in the parietal lobe. In this case there is in part a softening, which has destroyed in the right hemisphere, the region of the cortical visual center, in part a focus of softening located in the left hemisphere, which occupies the whole substance of the pli-courbe and the neighboring region of the temporal and parietal lobes. These observations confirm the results of observers as to the origin of intra-cerebral hemianopsia, as in these two cases,,in spite of the great extent of the lesions, degeneration of the fibers of Gratiolet and of the corpus callosum was not observed. The lesion then was strictly focal in its nature, in the optic tract.

SCHWAB.

KERNIG'S SIGNE (Kernig's Sign.) J. Roglet. (Journal de Médecin, Oct., 10, 1900.)

The author here reports on an extensive series of observations on the subject of Kernig's sign. This sign, as is well known, consists in the possibility of completely extending the leg on the thigh when in the sitting posture, in a patient suffering from certain meningeal diseases. There is a certain amount of contracture of the flexors of the leg. This contracture, on the other hand, disappears, and complete extension can be obtained when the patient is lying on the back. It is therefore easy to elicit this phenomenon if, after having ascertained the absence of all contracture in the supine position, the patient is made to sit up with his legs hanging free, or even sitting up in bed. Certain precautions are therefore necessary. The observer must see that the patient sits straight upright, and does not lean to one side or the other, and that the thighs form a right angle with the trunk, certainly not less. Disregard of this point may lead to considerable error. The attempt to obtain Kernig's sign is sometimes attended with considerable pain. The amount of extension obtained at the knee varies considerably. When Kernig's sign is well marked it is impossible to exceed a right angle; when slight, an obtuse angle equal to 135 degrees; but intermediate degrees may be obtained. Nor is the sign always bilateral; occasionally it has been observed on one side only, or to an unequal extent in both limbs. The intensity of the phenomenon may vary from day to day, or may even disappear completely. To be therefore sure of its non-existence several observations are necessary. Kernig's sign may appear at the same time as the other symptoms, rigidity of the neck, ocular symptoms, contracture, etc., but most usually it appears about the third or fourth day of the disease. In tuberculous meningitis its appearance is most delayed. It is very rare for it to be the only symptom present. Kernig's sign may disappear at variable periods. In meningitis ending fatally, it may persist up to the end, but in other cases it may disappear shortly before death, especially in cases, where owing to coma, there is general flaccidity, and all contractures give place to paralysis. It is under